Osteoarthritis

This lecture relied 100% on the textbooks (kumar and davidson) in covering the objectives alongside the doctor's notes.

Objectives :

- ★ Know the definition of osteoarthritis
- ★ Describe the pathophysiology of osteoarthritis
- ★ Learn the diagnostic work up of osteoarthritis
- ★ Know the differences between osteoarthritis and inflammatory arthritis
- ★ Learn treatment modalities available for management of Osteoarthritis

Editing file

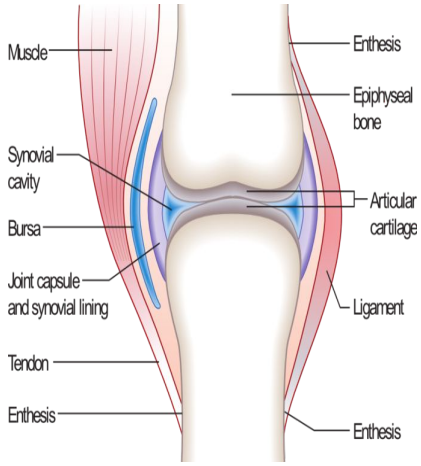
Color index

Original text
Females slides
Males slides
Doctor's notes 438
Doctor's notes 439
Text book
Important
Golden notes
Extra

Normal Synovial Joint structure

1 Articular cartilage

- **Hyaline cartilage** lining the bones within a joint.
- It is **avascular** and derives nourishment from synovial fluid.
- Articular cartilage (chondrocytes) surrounded by extracellular matrix includes proteoglycans and collagen.
- **The cartilage facilitates joint function and protects the underlying subchondral bone by:**
  - Distributing large loads
  - Maintaining low contact stresses
  - **Reducing friction at the joint.**
- **Defects in articular cartilage and underlying bone are features of osteoarthritis.**

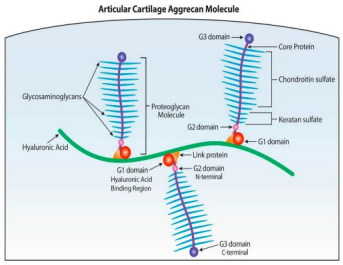


2 Joint capsule and synovial membrane

- The bones of synovial joints are connected by the **joint capsule**, a fibrous structure richly **supplied with blood vessels, nerves and lymphatics** that encases the joint.
- Ligaments are regional thickenings of capsule that act to stabilise joints
- **Synovial membrane** is the inner surface of the joint capsule
  - Comprising an **outer layer** (of blood vessels and loose connective tissue that is rich in type I collagen), and an **inner layer** (consisting of two main cell types. **Type A** synoviocytes are phagocytic cells, **type B synoviocytes** are fibroblast-like cells that **secrete SF**)

3 Synovial fluid

- Viscous liquid that lubricates the joint
- **Formed by synoviocytes:**
  - Synovial cells also manufacture **hyaluronic acid** (HA, also known as hyaluronate)
    - A **glycosaminoglycan** that is the major noncellular component of synovial fluid.



- **Its function:**
  - Synovial fluid supplies **nutrients** to the avascular articular cartilage.
  - Provides the **viscosity** needed to **absorb shock** from **slow** movements
  - Provides **elasticity** required to **absorb shock** from **rapid** movements

4 Subchondral bone (juxta-articular bone)

- The bone that abuts a joint
- Highly **vascular**
- Comprises a light framework of mineralized collagen enclosed in a thin coating of tougher, cortical bone.
- **It withstands pressure poorly if the normal articular hyaline cartilage is worn away, as in osteoarthritis**

Osteoarthritis خشونة المفاصل

Osteoarthritis

- **Heterogeneous group of conditions resulting in common histopathologic and radiologic changes involving Entire joint structures, including:** The articular cartilage, the subchondral bone and the synovium
- Osteoarthritis (OA), or **degenerative joint disease**, is a chronic, slowly, progressive, erosive damage to joint surfaces; this **loss of articular cartilage** causes increasing pain with minimal or **absent inflammation**

◄ Epidemiology :

- Internationally, osteoarthritis is the **most common articular disease**. Estimates of its frequency vary across different populations.
- 80-90% of individuals **older than 65** years have evidence of radiographic osteoarthritis.
- The prevalence of osteoarthritis is higher among **women** than among men.
- Interethnic differences in the prevalence of osteoarthritis have been noted.

◄ Risk factors

- **Age** (> 55 years)
- **Obesity** There's a strong association between obesity and OA, **particularly of the hip**. This is thought to be due partly to biomechanical factors due to cytokines released from adipose tissue
- **Hereditary**
- **Genetics**: The gene that encodes **collagen type II (COL2A1)** is a candidate gene for **familial OA** but there is no single gene that associates with all patterns of OA. COL2A1 is associated with **early polyarticular arthritis**
- Hypogonadism **Oestrogen appears to play a protective role**: lower rates of OA have been observed in women who use hormone replacement therapy (HRT), and women who receive aromatase inhibitor therapy often experience a flare in symptoms of OA.
- **Osteoporosis:** reduces risk of OA
- Gender: Polyarticular OA is more common in women; a higher prevalence after the menopause suggests a role of sex hormones
- **Trauma:** a fracture through any joint predisposes. Meniscal & cruciate ligament tears cause OA of the knee
- **Hypermobility:** increased range of joint motion and reduced stability lead to OA
- **Congenital joint dysplasia**
- **Joint congruity**
- **Occupation:** miners develop OA of the hip, knee, and shoulder, cotton workers OA of the hand, and farmers OA of the hip.
- **Sport:** Repetitive use and injury in some sports cause a high incidence of lower-limb OA

◄ Causes & Classification

Primary (Idiopathic)	• No identifiable underlying cause
Secondary (Known cause)	• **Preexisting joint damage:** Rheumatoid arthritis, Gout, Spondyloarthritis, Trauma , Septic arthritis, Paget's diseases, Avascular necrosis e.g. corticosteroid therapy and overuse/abnormal use(e.g. athletes) • **Metabolic diseases:** Cartilage calcification, **Hereditary hemochromatosis**[1], Acromegaly, Wilson disease, alkaptonuria • **Systemic disease**: Haemophilia-recurrent haemarthrosis, haemoglobinopathies, e.g. sickle cell disease, neuropathies • **Others:** Diabetes Mellitus, meniscectomy[2]

1- **It** is a condition that leads to abnormal iron deposition in specific organs. There are two main types: primary (hereditary) and secondary (e.g., transfusion-related).
2- The surgical removal of all or part of a torn meniscus.

Osteoarthritis خشونة المفاصل

◀ Pathogenesis[1]

- Consequent structural changes include **surface fibrillation** and **ulceration** with **loss of cartilage** that exposes underlying bone to increased stress, producing **microfractures and cysts** leading to abnormal **sclerotic subchondral bone** and **overgrowths** at the joint margins, called **osteophytes.**
- **Degeneration of articular cartilage**: is the defining feature of OA
- The destruction of joint tissues in OA is mediated by a variety of inflammatory mediators and proteases, including several matrix metalloproteinases (MMPs), and cysteine proteinases.

01

In early stages: **Swelling** of the cartilage usually occurs due to chondrocyte hypertrophy

02

The level of **proteoglycans** eventually **drops** very low, the cartilage softens and **lose elasticity** compromising joint surface integrity.

03

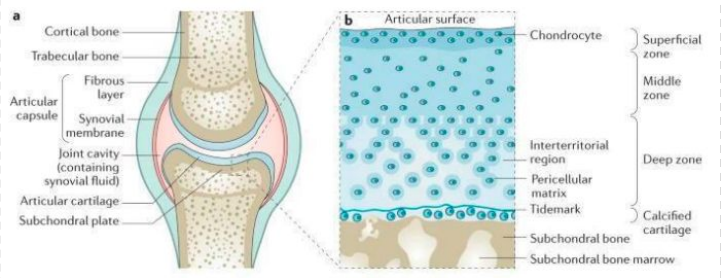
Fissures and **fibrillations**[A] (vertical clefts) develop along on the surface of an osteoarthritic joint

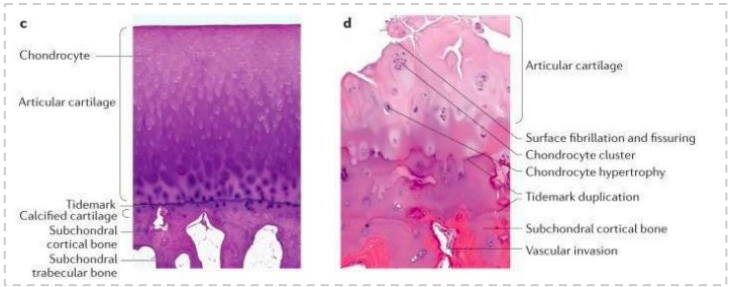
04

Over time , the loss of cartilage results in **loss of joint space** [2] and articulation of the bones (Advanced OA)

05

A **greater loss** of joint space occurs at those **areas experiencing the highest loads.**





◀ Bone changes

01 **Articulation:** Bone denuded of its protective cartilage continues to articulate with the opposing surface

02 **Stress:** Eventually, the increasing stresses exceed the biomechanical yield strength of the bone

03 **Eburnation**[3]**:** The subchondral bone responds with vascular invasion and increased cellularity, becoming thickened and dense (a process known as eburnation) at areas of pressure.

04 **Cystic Degeneration:** Subchondral bone undergo cystic degeneration

- Osteoarthritic cysts are also referred to as subchondral cysts, pseudocysts, or geodes and may range from 2 to 20 mm in diameter.
- Osteoarthritic cysts in the **acetabulum** are termed **Egger cysts**.

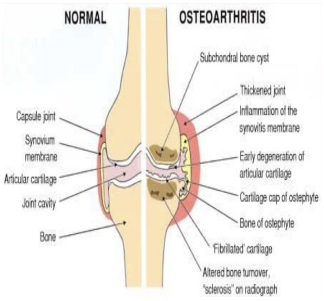
1- **In summary,** Chronic mechanical stress on the joints and age-related decrease in proteoglycans→ cartilage loses elasticity and becomes friable → degeneration and inflammation of cartilage → joint space narrowing and thickening and sclerosis of the subchondral bone

2-Calcium pyrophosphate and basic calcium phosphate crystals often become deposited in the abnormal cartilage

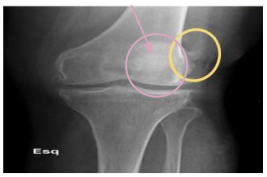
3- A degenerative condition of bone or cartilage characterized by unusual hardness and a polished appearance.

Osteoarthritis خشونة المفاصل

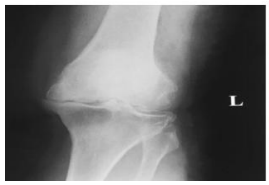
◀ Joint changes



- Vascularization of subchondral marrow
- Osseous metaplasia of synovial connective tissue
- Ossifying cartilaginous protrusions lead to irregular outgrowth of new bone **(osteophytes)**[1].
- Fragmentation of these osteophytes or of the articular cartilage itself results in the presence of intra-articular loose bodies **(joint mice).**



Early X-ray showing: Osteophytes, some narrowing and subchondral sclerosis



Advanced OA: The cartilage is lost, femoral epicondyle articulates directly with the tbital epicondyle and severe subchondral sclerosis

◀ Other changes

- **Synovium**:
  - Often hyperplastic and may be the site of inflammatory changes
  - **Osteochondral bodies** commonly occur within the synovium, reflecting secondary uptake and growth of damaged cartilage fragments.
- **Capsule**: The outer capsule also thickens and contracts, to retain the stability of the remodelling joint
- **Muscles**: The muscles surrounding affected joints commonly show **wasting**. (Because the person doesn't move)

◀ Osteoarthritis progression

Stage 1

Breakdown of the cartilage matrix occurs.

Stage 2

Involves the fibrillation and erosion of the cartilage surface

Stage 3

A chronic inflammatory response in the synovium.

Further progression

- The above events alter the joint architecture, compensatory bone overgrowth occurs.
- Joint architecture is changed
- Mechanical and inflammatory stress occurs on the articular surfaces, the disease progresses unchecked.

◀ Differential diagnosis

- Crystalline arthropathies (ie, gout and pseudogout)
- Inflammatory arthritis (eg, rheumatoid arthritis)
- Septic arthritis or post infectious arthropathy
- Seronegative spondyloarthropathies (eg, psoriatic arthritis and reactive arthritis)
- Fibromyalgia
- Tendonitis

1-Fibrocartilage is produced at the joint margin, which undergoes endochondral ossification to form osteophytes.

Osteoarthritis خشونة المفاصل

◀ Clinical presentation

★ The main presenting symptoms are **pain** and **functional restriction**

The pain is:

- Insidious onset **over months or years**
- Variable nature over time ('good days, bad days')
- **Worse with movement** and weight-bearing, **relieved by rest**
- **Brief (< 15 mins) morning stiffness**
- Brief (< 5 mins) 'gelling' after rest [1]
- Usually only one or a few joints painful

Clinical signs

- **Restricted movement** Due to capsular thickening or blocking by osteophyte
- Palpable, sometimes audible **coarse crepitus** due to rough articular surfaces (rough cartilage and bone rubbing together in a joint)
- Bony swelling around joint margins
- Deformity, usually without instability
- Joint-line or periarticular tenderness
- Muscle weakness and wasting
- Mild or absent synovitis

◀ Involved joints

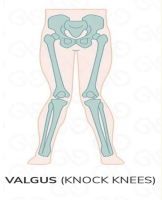
Weight-bearing joints

- **The knee** most common joint affected
- **The hips**
- Cervical and lumbosacral spine
- Feet

Non-Weight Bearing joints

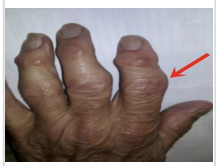
- The distal interphalangeal (DIP) more commonly affected compared to PIP
- The proximal interphalangeal (PIP) joints.
- **the carpometacarpal (CMC) joints.**
- The first metatarsophalangeal (MTP) joint of the foot.

Valgus deformity, less commonly. In a young lady : the knees will be close to each other **knock-knees (genu valgum),** usually seen in 2° Osteoarthritis (RA)

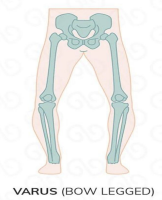


Bouchard's nodes
(Bony swelling at the **PIPJ**)
pain and nodular thickening on the dorsal sides of the **Proximal interphalangeal joints**

Suggests primary OA

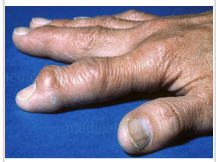


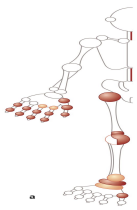
Varus deformity, Loss of cartilage usually begins medially "angle is outward". In older people: the knees will be away from each other we call it **Bow legs (or genu varum)** usually seen in 1° Osteoarthritis



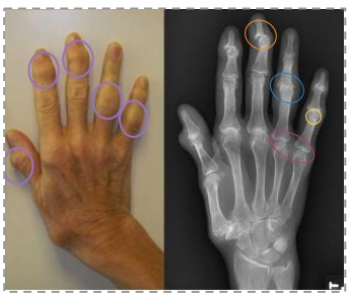
Heberden's nodes
(Bony swelling at the **DIPJs**)
pain and nodular thickening on the dorsal sides of the **distal interphalangeal joints**

Suggests primary OA





Red circles indicate the more commonly affected sites, and pale ones the less commonly affected sites.

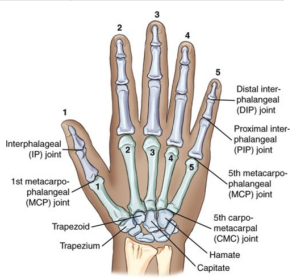


Normal space

subchondral sclerosis & osteophytes

Multiple subchondral cysts

Palmiflexion, can progress leading to dislocation



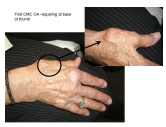
1- Condition that occurs when a joint has been at rest for too long and the synovial fluid becomes thickened , making movement difficult

◀ Types of osteoarthritis

Nodal OA

Characterised by:

- Pain, stiffness and swelling of **one or more PIP and DIP joints,** with DIPs being more often involved than PIPs. Typical pattern of polyarticular involvement of the hand joints.
- The inflammatory phase settles after some months or years, leaving painless bony swellings posterolaterally: **Heberden's nodes (DIPs) and Bouchard's nodes (PIPs)**
- Involvement of the **first CMC joint** is also common causing **squaring of the thumb** base



- Has Good long-term functional outcome for hands
- Predisposition to osteoarthritis at **other joints**, **especially knees** → **Generalised nodal OA**
- **Female preponderance**, Strong genetic predisposition, Peak onset in late middle age (around female menopause)

Knee OA

- Has a strong relationship with **obesity**
- Mostly **bilateral with symmetrical** involvement
- May be isolated or as part of generalised nodal OA of the hand in elderly women.
- Principally targets the patellofemoral and **medial tibiofemoral compartments** leads to a **varus "bow-legged" deformity**. But eventually spreads to affect the whole of the joint

Hip OA

Two major subgroups:

- **Superior-pole hip OA :**
  - Most common, usually affect men
  - **Unilateral** at presentation.
  - Affect the **upper surface of the femoral head** and adjacent acetabulum.
  - Has poor prognosis.
- **Medial cartilage (central) loss:**
  - Usually affect women, bilateral.
  - Associated with hand involvement (NGOA). Has better prognosis.

Spine OA

- Cervical (cervical spondylosis) and lumbar spine (lumbar spondylosis) are the most common targeted sites.
- Typical presentation is **pain localised to the low back** region or the neck, **relieved by rest** and worse on movement.
- May be complicated with spinal stenosis or nerve root compression → neurological signs and radiation of pain

Erosive OA

- This term describes an unusual group of patients with hand OA who have a more prolonged symptom phase, more overt inflammation, more disability and worse outcome than those with nodal OA.
- Distinguishing features include preferential targeting of PIP joints, **subchondral erosions on X-rays,** occasional **ankylosis**[1] of affected joints and lack of association with OA elsewhere.

1- Abnormal stiffening and immobility of a joint due to fusion of the bones.

Osteoarthritis خشونة المفاصل

◀ Investigations

Osteoarthritis is often diagnosed based on the patient's history and the presence of typical clinical features. Radiographic signs often do not correlate with the patient's reported symptoms or clinical findings; therefore, imaging is usually used to **support** the diagnosis

1 Plain Radiography (X-Ray)

- **The most accurate test of affected joint (1st line)**
- Only **abnormal when the damage is advanced**. They are useful in preoperative assessments.
- X ray changes do not correlate with symptoms, you might have significant x ray changes with no symptoms, and have severe symptoms with mild cray changes
- For knees, a standing X-ray (stressed) is used to assess cartilage loss and 'skyline' views in flexion for patellofemoral OA.
- **Findings include:** narrowed joint space(loss of joint space), Osteophytes (spurs of bone), Subchondral sclerosis (increase of bone density along joint line), and Cyst formation (subchondral cyst)

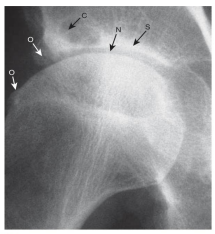


Fig. 24.21 **X-ray of hip showing changes of osteoarthritis.** Note the superior joint space narrowing (N), subchondral sclerosis (S), marginal osteophytes (O) and cysts (C).

Mnemonic of OA radiographic findings:
LOSS

Loss of joint space
Osteophytes
Subarticular sclerosis
Subchondral cysts

2 MRI, CT scan and US

- **MRI:** demonstrates meniscal tears, early cartilage injury and subchondral bone marrow changes (osteochondral lesions). MRI of spine should be done **if nerve root compression or spinal stenosis are suspected.**

3 Blood Tests

- Lack specificity; the ESR is usually normal, although high-sensitivity CRP may be slightly raised. RFs and antinuclear antibodies are negative.
- serology must be performed to identify whether it is primary or secondary OA

4 Arthrocentesis

- Joint analysis: **WBCs >200mm^3 and less than <2000mm^3**. Lymphocytes will be predominant since it is a chronic process.
- Keep in mind below 200 WBC is normal and higher than 2000 indicates inflammation.
- It is performed in suspicion of septic arthritis

5 Arthroscopy

- reveals early fissuring and surface erosion of the cartilage.
- may show a thickened capsule, synovial hypertrophy

Osteoarthritis خشونة المفاصل

◀ Management

Pharmaco-therapy

- Stem cell therapy
- There are no proven agents which halt or reverse OA.
- **Paracetamol (First line)** is effective and should be prescribed **before NSAIDs**. Addition of a topical NSAID, and then capsaicin, for knee and hand OA can be helpful.
- Oral **NSAIDs** should be considered in patients **who remain symptomatic**
- **Intra-articular corticosteroid injections** produce short-term improvement when there is a **painful joint effusion.**

1-Try to use topical before you use systemic medications:**Topical is better, why? to avoid the side effects.** for ex:

- Topical NSAIDs and it is **green** in ACR. (and it is recommended to be used)
- Topical capsaicin (from peppers) you feel like burning in your skin because it over stimulates the pain receptors then downregulate it> desensitization will occur.

2-Systemic medications: **IF TOPICAL IS NOT ENOUGH THEN YOU GO TO ORAL and try to use a medication with least side effects.**

for ex:

- Start the oral with acetaminophen (paracetamol) simple drug, low side effects.if the paracetamol is **not enough,** we use **Solpadeine.**
  Solpadeine: contains **codeine and paracetamol** (the codeine here is only 8mg so no addiction).
  if the **Solpadeine** is **not enough,** then we give NSAIDs.
  #NSAIDs two types :
  1-COX-2 inhibitor : if he have GI side effects we use it
  2-non-selective NSAIDs: if he doesn't have any risk we use it.

3-Intra-Articular Injections (IA) : -steroid-if the patient have only one joint and there is effusion-sign of inflammation-
4-other supportive drugs :(not recommended) ex.Glucosamine,chondroitin sulfate.
Duloxetine : used in depression

Non Pharmaco-therapy

- **Education**: Education of the individual about the disease and its effects reduces pain, distress and disability and increases compliance with treatment.
- **Lifestyle modification :**
  - Physical and rehab therapy Local strengthening and aerobic exercises improve local muscle strength, improve the mobility of weight-bearing joints and improve general aerobic fitness.
  - **Weight loss** It has a substantial beneficial effect on symptoms if the patient is obese and is probably **one of the most effective treatments** available for OA of the lower limbs

1- weight loss (if the OA was in weight bearing joints) reduction of weight is very effective to the the progression of OA -you cannot stop it-
2-exercise: For ex. knee (what are the muscles that you exercise it)? quadriceps (also retard the progresion of OA -you cannot stop it-)
3-using Cane (imp in elderly): -very helpful and strongly recommended- you hold the cane with the contralateral side (the healthy side).
4-orthotic braces: for ex. OA in neck use soft collar and don't use it for long time because it will cause muscle wasting.
5-massage and pulsed vibration therapy كلها خرطي بس عشان البزنس

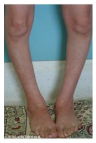
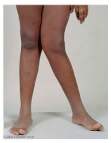
Surgery

- Arthroscopy (Arthroscopy for knee OA is not beneficial)
- Fusion and joint Lavage (Aka: Arthrodesis) (It is a surgical procedure that joins the bones together so, they no longer move or rub against each other)
- Osteotomy (Is any surgery that cuts and reshapes your bones. You may need this type of procedure to repair a damaged joint. It's also used to shorten or lengthen a deformed bone that doesn't line up with a joint like it should).
- Arthroplasty (Complete or partial replacement of a joint using an endoprosthesis)

Total joint replacement surgery is by far the most common surgical procedure for patients with OA. Surgery should be considered when there is significant impact on the quality of life despite optimal medical therapy and lifestyle advice.

Overview

How to differentiate between inflammatory and non inflammatory arthritis?(obj)

	Non inflammatory (degenerative)	Inflammatory
Character of pain	• Pain is worse in the evening • pain relieved at rest • **Pain increases with movement**	• Pain is worse in the morning • severe pain at rest • **pain is relieved with movement**
Posture	Genu varum "seen in primary"  Genu Varum "Bow-legged"	Genu valgum  Genu Valgum "Knock-Knee"
Degree of stiffness	Morning stiffness <30 mins	Morning stiffness >60mins
Signs of inflammation	No cardinal signs of inflammation	Cardinal signs of inflammation (redness, hotness, swelling)

★◀ Osteoarthritis VS Rheumatoid arthritis (obj)

	Osteoarthritis	Rheumatoid arthritis
Pathogenesis	Mechanical - **wear and tear** destroys articular cartilage **(degenerative joint disorder)**	Chronic, systemic **autoimmune** disease
Predisposing factors	Major risk factor is **age** (common after 60 years); additional risk factors include **obesity** and **joint trauma.**	Associated with **HLA-DR4**, classically arises in women of late childbearing age
Presentation	- Pain and functional restriction ★ **Early morning stiffness** (lasting **LESS** than 30min) - targeting the hips, knees, PIP and DIP joints, neck and lumbar spine - But **doesn't involve MCP** - Asymmetric joint involvement - **No systemic symptoms**	- Pain and joint swelling - **Early morning stiffness** (lasting **MORE** than 1hr) that gets **better with movement** - Targets small joints of the hands, feet and wrists. And there is large joint involvement - There's spindling of PIPJs and MCP but NOT DIPJs or 1st CMC - Symmetric joint involvement - **Systemic symptoms** - Extra-Articular manifestations
Joint findings	- **Osteophytes** (bone spurs) - Joint space narrowing, Subchondral sclerosis and Cyst formation - **Heberden** and **Bouchard nodes**	- **Pannus** (proliferative granulation tissue) - Erosions, cervical subluxation and ulnar finger deviation - swan neck and boutonniere deformities

Summary

	Osteoarthritis
Pathogenesis	Mechanical - **wear and tear** destroys articular cartilage **(degenerative joint disorder)** Gradual destruction of the joint through the loss of articular cartilage
Predisposing factors	• Age • Obesity • Heredity • Gender (women) • Hypermobility • Osteoporosis • Trauma to joints • Congenital joint dysplasia • Joint congruity • Sport (Repetitive use & injury)
Presentation	- Pain & functional restriction - Early morning stiffness (lasting LESS than 30 min) - Asymmetric joint involvement
Investigations	most accurate : X-ray All labs are normal
Treatment	- Lifestyle modification (weight reduction & rehab) - NSAIDs

#Take home message from Doctor:
-Try to use topical before you start systemic.
-paracetamol is a very good drug,not enough go for Solpadeine, not enough go for NSAIDs.
-Do not forget exercise, don't forget reduction of weight, and using of orthotic devices(cane).

Take home Messages

- **Heterogeneous group of conditions resulting in common histopathologic and radiologic changes involving Entire joint structures, including:** The articular cartilage, the subchondral bone and the synovium
- **Hereditary hemochromatosis is a secondary cause of OA**
- **Bouchard's nodes** (Bony swelling at the **PIPJ**) **"suggests primary OA"**
- **Heberden's nodes**(Bony swelling at the **DIPJs**) **"Suggests primary OA"**
- **Valgus deformity,** less commonly.
- **Varus deformity,** Loss of cartilage usually begins medially "angle is outward". **Bow legs (or genu varum)**
- **Brief (< 15 mins) morning stiffness**
- **Pain is worse with movement** and weight-bearing, **relieved by rest**
- **Paracetamol (First line)** is effective and should be prescribed **before NSAIDs**. Addition of a topical NSAID, and then capsaicin, for knee and hand OA can be helpful.

Lecture Quiz

Q1: A 75-year-old woman presents to accident and emergency complaining of pain in her knees. She mentions that this has been troubling her for several months. Pain is generally worse in the evenings and after walking. On examination, there are palpable bony swellings on the distal interphalangeal joints of the fingers on both hands. In addition, there is reduced range of movement and crepitus in the knees. What is the most likely diagnosis?

A- Rheumatoid arthritis
B- osteoarthritis
C- reactive arthritis
D- polymyalgia rheumatica
E- gout

Q2: A 79-year-old woman presents to her GP with pain in the left knee. This is particularly bad in the evenings and is stopping her from sleeping. The GP explains that her discomfort is most likely due to osteoarthritis and arranges for her to have an x-ray of the knee. Which of the following descriptions are most likely to describe the x-ray?

A- Reduced joint space, subchondral sclerosis, bone cysts and osteophytes
B- Increased joint space, subchondral sclerosis, bone cysts and osteophytes
C- Reduced joint space, soft tissue swelling and peri-articular osteopenia
D- Increased joint space, soft tissue swelling and peri-articular osteopenia
E- normal x-ray

Q3:A 67-year-old woman comes to the physician for the evaluation of bilateral knee pain for the past year. She reports that the pain is worse with movement and is relieved with rest. She has type 2 diabetes mellitus. The patient says her mother takes leflunomide for a "joint condition." The patient's medications include metformin and a multivitamin. She is 165 cm (5 ft 5 in) tall and weighs 85 kg (187 lb); BMI is 31 kg/m$_2$. Vital signs are within normal limits. Physical examination shows pain both in complete flexion and extension, crepitus on joint movement, and joint stiffness and restricted range of motion of both knees. X-ray of the knee joints shows irregular joint space narrowing, subchondral sclerosis, osteophytes, and several subchondral cysts. There is no reddening or swelling. Which of the following is the most appropriate pharmacotherapy?

A- Intra-articular glucocorticoid injections
B-Administration of ibuprofen
C-Administration of infliximab
D- Administration of celecoxib
E- Administration of oral prednisolone

Q4: A 52-year-old woman comes to the physician because of a 4-month history of progressive pain and stiffness of the fingers of her right hand that is worse at the end of the day. She works as a hairdresser and has to take frequent breaks to rest her hand. She has hypertension, for which she takes hydrochlorothiazide. Two weeks ago, she completed a course of oral antibiotics for a urinary tract infection. Her sister has systemic lupus erythematosus. She drinks one to two beers daily and occasionally more on weekends. Over the past 2 weeks, she has been taking ibuprofen as needed for the joint pain. Her vital signs are within normal limits. Physical examination shows swelling, joint-line tenderness, and decreased range of motion of the right first metacarpophalangeal joint as well as the 2nd and 4th distal interphalangeal joints of the right hand. Discrete, hard, mildly tender swellings are palpated over the 2nd and 4th distal interphalangeal joints of the right hand. Which of the following is the most likely underlying mechanism for these findings?

A- Degenerative disease of the joints
B- Autoimmune-mediated cartilage erosion
C- Monosodium urate crystal precipitation in the joints
D Bacterial infection of the joint space
E- Calcium pyrophosphate dihydrate crystal precipitation in the joints

Answers: Q1:B | Q2:A | Q3:B | Q4:A

Answers Explanation File!

GOOD LUCK !

This work was originally done by ***438 Medicine team:***

Team Leaders

- *Raghad AlKhashan*
- *Amirah Aldakhilallah*
- *Mashal AbaAlkhail*
- *Nawaf Albhijan*

Member : Ghada Alsadhan, Razan alzohaifi, Amirah Aldakhilallah, Deana Awartani

Note taker : Sarah Alhelal

Edited by ***439 Medicine team:***

Team Leaders

- *Shaden Alobaid*
- *Ghada Alabdi*
- *Hamad Almousa*
- *Naif Alsulais*

Member : Rima Alomar

Note taker : Ahmed Alhawamdeh

CONTACT US THROUGH OUR EMAIL :
MEDICINE439@GMAIL.COM